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Featured Article:

Chronic Bodily Pain, the Dark Side of Neuroplasticity and New Experiential Approaches to Active Treatment:

The Role of Activating *Complex Sensations, Newly Differentiated Actions*, and *Enlightened Outlooks* as Competing Behavioral Pathways for Perpetuating Sustainable Change in Persons with Chronic Pain

By

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On any given moment, the state of the body is a reflection of the state of the nervous system - including somatosensory, cognitive, visceral, neuroendocrine and immune systems; as well as our own individual biopsychosocial systems - as an extension of it all. These processes are continually updated by our quality of our somatosensory experience.

"In general, all forms of pain are affected by many high central nervous system activities."

INTRODUCTION:

Chronic Recurrent Musculoskeletal Pain Syndromes--whether *specific and localized* or *widespread and diffuse*…mildly burdensome or completely incapacitating -- still remain a common and difficult problem that tends to remain unresponsive or resistant to many varied kinds of treatment approaches-and often with only temporary suggestive results at best.

Implicit assumptions and beliefs about persistent pain in traditional clinical settings include the attribution that a *structural lesion* or a *focal tissue pathology* must always the be the cause, that pain correlates to extent and severity of actual tissue damage, and that chronic pain is in fact an implication that the tissues involved are either perpetually diseased or not healed; and / or an indication that some type of mechanical trauma must still be happening. A continued idea is that pain signals are transmitted only through peripheral nociceptors -due to a direct input of stimulus arising from the provocation of localized damaged tissues--and that these signals occur along a predictable network of telephone wires via simple excitation and a one-way direction of conduction - *en route* from the body to brain - in order to alert the brain.

The procedural outcome of such structurally-determined beliefs has resulted in a history of interventions that involve simply *removing the painful part* -- even to the extent of removing 294 sq.cm of 'painful skin' from the thoracic region of a patient diagnosed with post herpetic neuralgia -- as late as October, 2000--but only to find that pain pathology significantly worsened to actually exceed pre-surgery levels, and with altered distribution of expanded symptom topography at 5 year follow-up-- this despite a brief initial period of symptom relief and despite full healing of original lesion(s).

The fact is that *tissues heal* -- typically fully well within 1- 3 months --yet *pain can persist for many months -- and into many years*.

In addition, a growing number of individuals commonly acquire painful musculoskeletal conditions without traumatic injury. Pain can be represented quite vividly in amputated limbs that no longer exist (phantom limb pain) and likewise, spontaneously exist in the absence of a provoking stimulus (central sensitization / allodynia / central neuropathic pain) in the same manner that *ringing of the ears* occurs in the absence of an auditory stimulus (tinnitus).

Could it be that the brain has become hyper-alert through conditioned sense memory -- and is now alerting the body of an anticipated or expected threat through maintaining a pain signaling pathway selection bias-- real, virtual, inadvertent, acquired or otherwise?

THE ROLE OF BRAIN NEUROPLASTICITY and the CO-CONDITIONING of Chronic Pain States:

Ultimately, all pain is the net output of constitutive interpretation and affective experience being generated from multiple influences *to, from, and throughout the brain*.

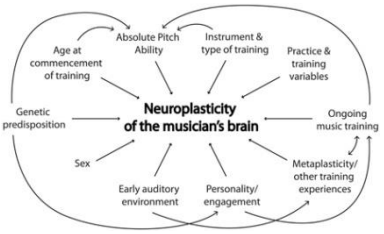
A change in the *internal processing of pain signaling* (including representational cognitive processes / emotional limbic association, and mapping of constructs throughout the body) is ultimately *experience dependent* and leads to corresponding changes in the neuronal selection and inhibitory / excitatory functions within the brain and spinal cord. Existing genetic factors co-conspire with experience-based epigenetic, gene expression opportunities to select and constitute toward a particular neuropeptide profile and a corresponding constructed pathway of reflective distribution that becomes intrinsically reinforced and co-conditioned to maintain its acquired state or mapping of familiar conditions (i.e. learning); and can thereby behave largely independent (as a *virtual body* on its own) of actual nociceptive input from the periphery (i.e. Information from tangible and actual body parts via usual afferent & efferent pathways).

In this, we can perhaps now re-situate chronic pain syndromes to be re-interpreted or re-classified --not as a peripheral pathology representative of *actual* tissue damage far away

within the body that *needs to be fixed* --but rather as a mal-adaptive acquisition of inadvertent central bias within synapses and pathways, functionally occurring through conditioned processes and constructs involving self-reinforcement and learning -- thus developing into a central ***plasticity disorder***.

Hebb's Axiom states that *'That which fires together - wires together'* (Hebb's Law, 1949) and this rule of process for encoding our everyday experience seems to apply even in cases where involuntary and undesirable or unpleasant *approach-avoidance* phenomena can occur- *as is the case with continued pain.* These contingencies can *and in fact do* contribute toward an unintended pathway construction of brain-body representations that have become somehow skewed and disordered from our normative, everyday proactive function; constituting instead the acquired development of a semi-conscious, protective matrix of learned chronic pain.

Thus, the uncanny aspect is that plasticity's known mechanisms involving biased synaptic efficacy, excitatory protein synthesis, and re-routing of selected information to widespread areas of the brain --many of which are known to be involved in both cognitive-perceptual and emotional function; but in shared relationship with pain processing also ---all happens at a level of *involuntary involvement*; plastic changes co-occurring on multiple levels (firing together), and evolving into actual morphologic changes and constructs (wiring together) at a level well below our ordinary conscious awareness. We are all products of multi-systemic interactive conditioning -- which ordinarily allows us to process information and to function better.



But by now, it is well known from evidence in neuroscience that the human brain undergoes neuroplastic cortical reorganization –distorted representations in the mapping of body schema and the corresponding dexterity of the body—in response to sensitizing or challenging experiences—but especially with chronic pain. (Flor, Mosely, Wand, Doidge). Furthermore, re-constructive and de-constructive body maps / virtual homunculi are being constantly updated and dynamically sculpted by ongoing interoceptive and exteroceptive experiences--both habitual and non-habitual-- and existing at multiple levels throughout the entire CNS including cerebellar, insular, thalamic and associated limbic regions.

Training Complex Sensation & Newly Differentiated Actions as a Means to Develop Competing Neuronal Pathways: A Prospective Solution to Learn to Move without Bodily Pain?

Aversive events and chronic pain states commonly result in and/or co-contribute to disordered functioning of ***working body schema*** (i.e. sensory-motor deficits in the clarity, resolution, dexterity of everyday actions) correspondent with topographic disruption (smudging and dissociation) of *body-related* cortical representations --aka *the dark side of neuroplasticity*. And "despite intact / normal 'standard' neurological screening (i.e. pinwheels, reflex hammers, sharp-dull tactile thresholds and the like), *complex sensations* (graphesia, tactile acuity , visual-spatial body awareness, cross-modal representations, discriminative proportions, etc.) remain otherwise impaired - and are co-related to deficits in motor control."

It thus follows that due consideration is now necessary for exploring and attending to new Rx interventions which aim to train / entrain / re-train / and to **clarify complex sensations** -with the underlying assumption that "*accurate body perception underpins skilled movement, sensation, localization, laterality discrimination, cohesive emotional states, self-awareness, etc.*" --and that these competing sensations; their novel processes for re-routing & neuronal selection, and their corresponding emergent perceptions -- are all antithetical to the recurring and complex phenomenology of conditioned states of ongoing pain perception.

Tim's Take: *Some Added Thoughts & Summarizing Points*:

- *On any given moment, **the state of the body** is a reflection of **the state of the nervous system** - including neuroendocrine and immune systems --as well as bio-psychosocial systems as an extension of it.*

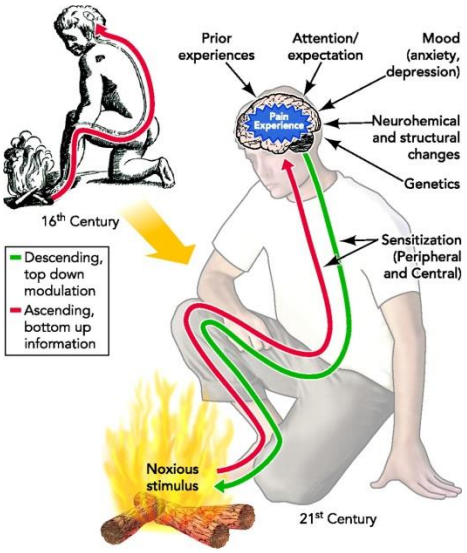
- These processes are continually updated by our quality of our somatosensory experience.

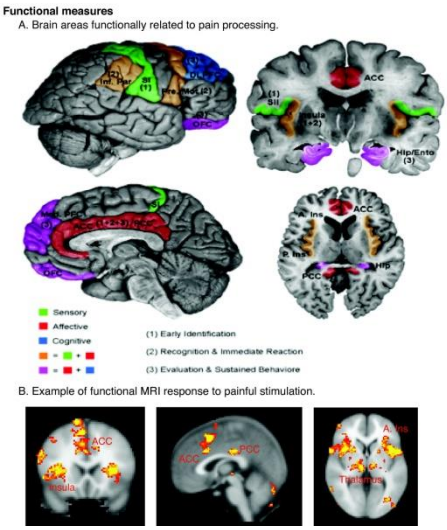
- **Notable quote from Roger Sperry, Ph.D., winner of the Nobel Prize for Medicine for his brain research, 1981:**

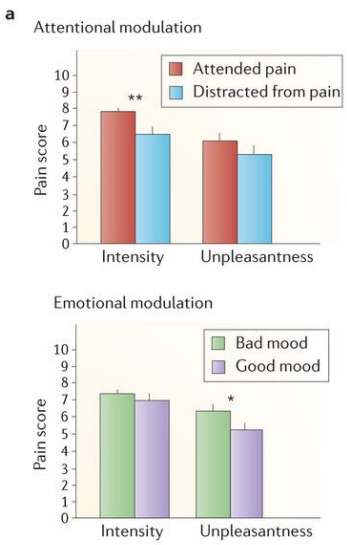
"*Better than 90 percent of the energy output of the brain is used in relating the physical body in its gravitational field. The more mechanically distorted a person is, the less energy available for thinking, metabolism, and healing*."

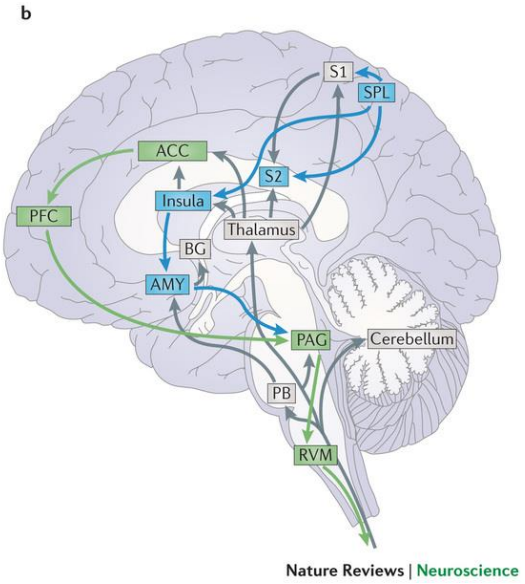
(Case in point: declinations in gait quality are correlated to incidence and prevalence of cognitive decline and is physically co-morbid as a functional presentation marker in persons with early onset dementia).

- Areas of the brain involved in pain processing of sensory vs. nociceptive, inter-association, and kinesthetic effector inputs & outputs can include: amygdala, hippocampus, anterior & posterior cingulate cortex, orbital frontal cortex, thalamic pathways, primary and secondary somatosensory cortex, supplementary and pre-motor cortex areas, primary motor cortex, pre-frontal cortex, insular cortex, posterior parietal complex, basal ganglia, cerebellum, and hypothalamus / HPA axis to ANS. Spinal cord and dorsal horn gating mechanisms.









"In general, all forms of pain are affected by many high central nervous system activities (i.e. descending influences).....and....it is known that electrical stimulation of pre-motor areas can suppress pain." (Moller, 2013).

- Training of high sensation, its corresponding organization, and effective action of its effectors in the real (or imagined) world --actually matters as a method of treatment outcome.

- *"We cannot make sense of what we have no sense of "* - Frank Wildman, Ph.D.

- "The body sculpts the brain's meaning" - Steven Schmidt, PT

- Cortical Somato-sensory maps are updated regularly by somato-sensory input and closely related to perception of self. Other methods have been devised to clarify vs. correct such erroneous mapping of the body -- most notably mirror box therapy and virtual realities --as well as *sensory-discriminative* approaches like the Feldenkrais Method.

- International colleague and fellow movement therapist practitioner, **Aurovici Sercomanens, D.O.** poses that the gate control theory / the phenomenon of sensory gating (Melzack and Wall, 1962) may be one of the primary ways as how both informative *hands-on* and slowly, gently applied *facilitated movement discrimination* sessions work from the outset. He describes it nicely here:

"Sensory gating means that the processing and perception of sense information is reduced by *the presence of other competing sense information.* If your nervous system is busy trying to process signals resulting from (constructed , synergistic) movement (sequences) you are making, or from the sensation of (informed and communicative) touch; it will have less ability to perceive and process pain signals, and hence, your pain will reduce."

Areas of *sensory gating* , *attention -perception modulation* , and *movement response selection* can exist at many & varied levels of the nervous system, and resultant perception is modulated by interactions between different neurons. "What the brain receives are nociception (receptor) signals and it decides how to interpret these and make them result in pain or not. Pain is the brain's output after interpreting the signals. So ultimately, Pain is an output (perception) from the brain, not an input from the body."

Sercomanens, 2013

- **Sensory Information at the Cross-Roads: *Differentiating the Thalamus* & *Inviting the Insula***:

  A more central neurophysiological rationale as to how uniquely constructed and *inquisitively slow* movement discrimination interventions might work much differently than other common procedural PT fitness & exercise *rapid repetition* routines; involves comparing and contrasting the competing levels of *sensory-relevant* information at more differentiated levels of thalamic processing:

  A) ……The **ventral thalamus** is wired to process sensory information with great accuracy and refined clarity of detail and to relay extracted, continually updated information to both *primary sensory* and polymodal *associative,* pre-frontal task-responsive cortices. These systems have been classically described as the "***Slow and Accurate***" systems. (The more discriminative mapping as to the 'Where' of it all as a "high route" of highly -processed appraisal before reaching inhibitory processes to the amygdala - thus with greater bias & potential for optimizing the positive regenerative outcome of descending modulation).

  In contrast:

  B) ……The **dorsal--medial thalamus** bypasses the primary cortices and instead sends axons directly to many dispersive areas of the brain, most notably secondary association cortices, limbic system nuclei, lateral nuclei of the amygdala, anterior cingulate, hippocampus, as well as PAG (periaqueductal gray) and reticular formation / activation. This part of the sensory system (non-classical or lateral, extra-lemiscal spinal-thalamic tract) is sub-cortically diffuse, less accurate and less detailed and has been described as "***Fast and Dirty***." (The more immediate 'What' ('the Hell') of it all as a "low route" of lesser-processed, reactive alarm response amplification to the amygdala; triggering an endocrine, autonomic, behavioral cascade that can thus be perceived vaguely as a state of immediate danger or threat in the background -- and co-associated with amplified pain signaling --but lesser discriminative body awareness in the foreground).

Complex Mobility Training and Perceptual Acuity approaches seek to construct experiences which conductively select a bias toward taking the neurological 'high road' through the ventral thalamic pathways via its emphasis on slow, non-threatening, highly discriminative and naturalistic movement synergies that have both informed and constituted our original sensory-motor development in gaining a clear and accurate, functional familiarity with our immediate world.

This serves as a competing stimulus in contrast to usual experience -- as with mindless repetition with weights and of working against everyday limitation with more and more effort - as is the usual mode of traditional, prescriptive, athletic-callisthenic oriented or 'corrective' styles of bodywork and physical therapy.

The Role of the Insula

The **Insula cortex** is involved in complex relationships between thalamic and amygdaloid nuclei, is anatomically convergent between orbital-frontal, secondary somatosensory, temporal, and parietal lobes and mediates vast assortments of information processing between sensory perception and cognitive function, emotions and motor control, temperature regulation, vestibular and autonomic homeostasis, self-awareness -- and especially *interoceptive awareness*, and sense of ownership; identification of 'one's own body'-- of which the phenomenology of individual pain experience is quite significant:

"There is both anatomical and some functional evidence of involvement of the insular lobe in the primary integration of multi-modal sensory input -- inclusive of (substrates for) pathological (chronic) pain, ***but so far, no treatment methods have been developed that target the insula."*** **(Moller, 2013).**

Though perhaps, some newer pilot study interventions aimed to integrate uniquely novel and functionally relevant sensory-motor experiences, developed through cross-modal representations of body perception, by utilizing visual-tactile acuity models to clarify hidden relationships, with a comprehensive intent toward clarifying an improved background working body schema; further aided by the emotional assurance to experience something refreshingly different from the usual -but altogether safe… might be a nice and welcoming first step…
…(whew)…

….*and this is the aim of the design approach within our prospective comparative efficacy study*!

- Outside of *Body Mind Centering* (BMC), some components of *The Franklin Method*, our first two-years of organic development during infancy, and perhaps Aikido practice, I know of no other comprehensive approach that so effectively entrains and co-conditions ***higher complex sensations*** together with ***implicit everyday movement*** and the ***corresponding development of higher integrative cognitions*** than the comparative approaches that we are currently investigating and researching in our study.

  Not usual callisthenic Yoga routines, not repeated Tai Chi, not usual Therapeutic Exercises, not even customary (sit still) deep meditation practices, and certainly not common fitness industry workout routines using redundantly predicable, sagittal plane biased weight machines, nor balancing precariously upon unstable physioballs, reformer platforms, isolated stretching techniques, regimented rubber band contraptions, and the like -- all of which are considered "*extra-functional*" in the scope of everyday life tasks and adaptive skill set preparedness.

This is a strong statement of assertion overall -- but it deserves to be heard, validated, and especially--researched!!!

Other Closing Thoughts:

- **Think Pathways and Matrices - not Parts**. "Pain is a multiple system output activated by an individual's specific pain neural signature. The neural signature (or neural tag) is activated whenever the brain concludes (perceives) that body tissues are in danger and action is required." (Moseley, 2003).

- Think ***Possibility model*** (something is inherently right-- just not discovered yet) in lieu of resorting to an accustomed ***Pathology model*** (something is inherently wrong and needs fixing) -- especially in the absence of life-threatening pathology.

In Closing -- An Opening!:

Research has shown that -- that despite best intentions -- many current treatment options have had deleterious effects on the health and well-being of persons afflicted with chronic pain - and in most cases, thereby reinforcing signaling pathways being cultivated towards the ever-protective and vigilant *dark side of neuroplasticity* -- thus perpetuating a mal-adaptive illness.

As our comparative efficacy research seeks to employ a newer active treatment model, we furthermore hope to develop and foster broader awareness of treatment interventions that are geared toward cultivating and optimizing the *lighter side of applied neuroplasticity.*

In this, we hope you will consider referral of your patients with chronic- recurrent neural-musculoskeletal pain as a *first line* consideration (if not also a moral imperative) towards the restoration of bodily resilience and sustainable change….in manners that afford a more comfortable quality of life for those afflicted, and an overall improved manner of feeling, of functioning, of contribution, and of being more fully human in the world. Thank You.

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